Original Article

Treatment of Shivering After Epidural Lidocaine

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Abstract: The effectiveness of intravenous meperidine and warm local anesthetic for prevention of postanesthetic shivering was evaluated in urology patients undergoing epidural blockade for extracorporeal shockwave lithotripsy. When administered before the blockade, meperidine, 12.5 mg or 25 mg, was not significantly better than saline placebo for preventing postepidural shivering. Changes in the concentrations of catecholamines or lidocaine did not result in differences between patients who shivered and those who did not shiver. In a second experiment, patients receiving body-temperature or room-temperature epidural lidocaine did not differ with respect to the incidence of postanesthetic shivering, onset of sensory blockade, or core temperature during a 30-minute observation period. The authors concluded that neither meperidine, in doses employed, nor body-temperature lidocaine prevents shivering after epidural blockade. This shivering appears to be different from that observed during emergence from general anesthesia. [Key words: Anesthesia, Epidural, Shivering, Local anesthetics, Lidocaine, Narcotic analgesics, Meperidine] Regional Anesth 1989;14:13-18.

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Postanesthesia shivering is known to increase ventilation and cardiac output, which can result in morbidity for patients with limited cardiopulmonary reserves. In addition, we have observed that shivering may cause muscle artifacts that trigger the Dornier shockwave generator inappropriately, and thus have the potential for inducing cardiac dysrhythmias as well as complicating the radiographic imaging of the renal stones.[1-3]

There are many treatments for postanesthesia shivering. Intravenous meperidine is reported to be effective for shivering after general anesthesia,[4-7] and warmed local anesthetic solutions may arrest shivering in some parturients after epidural blockade.[8,9] Shivering has been a relatively common occurrence among patients undergoing extracorporeal shockwave lithotripsy (ESWL) in our institution. We conducted a randomized prospective study of two treatment protocols: intravenous meperidine and body-temperature epidural lidocaine in urology patients receiving epidural anesthesia for ESWL.

Methods

Meperidine Prophylaxis

After approval from our institution's Human Investigation Committee, 60 unpremedicated ASA 1 and 2 patients scheduled for ESWL gave written consent to participate in an evaluation of the effects of intravenous meperidine on shivering after epidural blockade. On arrival in the anesthesia induction area, a venous heparin lock and separate intravenous catheter were placed. Blood samples were collected and stored to determine baseline catecholamine levels. Volume loading was begun with 500 ml of room-temperature lactated Ringer's solution with 5% dextrose. Approximately 15 to 30 minutes before placement of the epidural blockade, one ml of

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an unknown preparation was administered intravenously from previously prepared multidose vials labeled and coded by the hospital pharmacy. The three coded vials contained 12.5 mg/ml meperidine, 25 mg/ml meperidine, or saline placebo. The code was not broken until the entire study was completed. Each patient was placed in the lateral decubitus position, and the epidural space was identified at either the L2-3 or L3-4 interspace by the loss of resistance method. Approximately 20 ml of room-temperature lidocaine (1.54% with 1:220,000 epinephrine and 0.9 mEq $NaHCO_3$/10 ml) were injected. After a test dose, the epidural catheter was secured before the patient was repositioned. Additional lidocaine was administered in small aliquots at five-minute intervals, as needed, to establish a T5-7 sensory blockade to pinprick within 30 minutes. All patients were covered by a blanket that was only removed to assess the spread of the sensory blockade. Warming lights were not used. Shivering was diagnosed by a nurse previously trained to identify the clinical features of shivering, but unaware of the experimental protocol being used for each subject.

Blood samples were collected from a peripheral vein for assay of catecholamine levels after the patient arrived in the lithotripter induction area and again at the onset of shivering. Blood was collected approximately 15 minutes after epidural blockade from those patients who were not shivering. All blood was collected in prechilled tubes and stored for later assay by high-pressure liquid chromatography. Blood samples for lidocaine were obtained as soon as the patient shivered, and assayed using the fluorescein polarization immunoassay (TDx) method.

Body-Temperature Lidocaine

In the second portion of this study, another 26 unpremedicated ASA 1 and 2 patients scheduled for ESWL gave written consent to participate in a protocol evaluating the effects of body-temperature epidural lidocaine on shivering. Patients were randomized according to the last digit of the hospital number to receive either room-temperature (21-22°C) or body-temperature (37°C) lidocaine. After arrival in the induction area, patients received 500 ml of warmed (32-34°C) Ringer's lactated solution without dextrose and were positioned. The epidural space was identified using loss of resistance at the L2-3 or L3-4 space. Patients assigned to the body-temperature lidocaine group had their drug prepared in the following manner: Vials of lidocaine with epinephrine and vials of sodium bicarbonate were placed in a warm (43°C) water bath and allowed to equilibrate. Sodium bicarbonate was added to the lidocaine for a final concentration of 1.8% with 1:220,000 epinephrine and 0.9 mEq $NaHCO_3$/10 ml. After identification of the epidural space with saline, the warmed bicarbonated lidocaine solution was drawn up into a plastic syringe and injected through the Tuohy needle. The temperature of the solution was confirmed by fitting an inline temperature probe (Mon-a-therm 7000, St. Louis, MO) to the Tuohy needle and measuring injectate temperature in selected patients. Room-temperature lidocaine was prepared using lidocaine and sodium bicarbonate that had been stored in the induction room. The volume of injectate was standardized for body height in an effort to provide sufficient quantity of local anesthetic to obtain a T5-7 sensory blockade from a single injection. Patients less than 60 inches tall, between 60-66 inches, and taller than 66 inches received doses of 15, 20, and 25 ml, respectively. After a 3-ml test dose, the calculated volume was injected through the Tuohy needle over 90 seconds, the catheter was placed, and the patient was repositioned. Patients were covered with a thin blanket after injection. Sensory levels, core (tympanic membrane) temperature, and shivering were assessed for a 30-minute period by a trained nurse who was unaware of the injectate temperature. Shivering was graded as either mild or severe. Additional lidocaine was given if necessary, to obtain a T5-7 sensory blockade only after concluding the 30-minute observation period.

Statistical analysis of the results for these two protocols was performed using chi-square, Fisher's exact tests, and unpaired *t* tests.

Results

Meperidine Prophylaxis

Eighteen of 60 patients (30%) in this protocol shivered after epidural blockade. Men accounted for 60% of the ESWL population. However, men and women had identical rates of shivering. Although the results did not reach significance ($p = 0.42$), there was a trend suggesting that pretreatment with meperidine may reduce the incidence of postepidural blockade shivering (Table 1). Shivering began an average of 13.5 minutes after the epidural blockade was placed in 17 of 18 patients (94%), at which time the mean sensory level was T6.

There were no significant differences in either catecholamine or lidocaine levels between shivering and nonshivering groups (Table 2). Plasma concentrations of epinephrine were significantly elevated after epidural blockade irrespective of whether shivering had occurred.

Body-Temperature Lidocaine

The 26 patients were randomized into a body-temperature ($n = 13$) or a room-temperature ($n = 13$) epidural lidocaine group. The two groups were similar with respect to total volume of local anesthetic solution. Three patients (19%) in each group did not ob-

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TABLE 1. *Meperidine to Prevent Shivering*

Treatment	Total no. of Patients	No. Shivering	% Shivering
Placebo	20	8	40
Meperidine 12.5 mg	19	4	21
Meperidine 25 mg	21	6	28

Chi-square 1.697; $p = 0.42$.

TABLE 3. *Warmed Lidocaine to Prevent Shivering*

Treatment	Total no. of Patients	No. Shivering	% Shivering
21°C Lidocaine	13	6	46
37°C Lidocaine	13	4	31

$p = 0.34$ (Student's t test).

tain a T5–7 sensory blockade within 30 minutes of injection of epidural lidocaine. The incidence of shivering was not statistically different (p = 0.34) between the body-temperature (4/13) and the room-temperature (6/13) groups (46% vs. 31%) (Table 3). The mean time to first observed shivering was similar (11 vs. 15 minutes), as was the intensity of shivering.

The temperature of the injectate did not influence the onset of sensory blockade (Fig. 1). There was a trend suggesting that core body temperature in the room-temperature group (p = 0.08 at 5 minutes) was higher than in the body-temperature lidocaine group (Fig. 2).

Shivering patients did not differ from those who did not shiver with regard to time of onset of sensory blockade (Fig. 3). There were no differences in core body temperature between patients who shivered and those who did not shiver (Fig. 4).

Discussion

The overall incidence of shivering after epidural blockade for both protocols in this study was 32%. In the meperidine protocol, patients given a placebo before epidural blockade had a 40% incidence of shivering. In the body-temperature lidocaine protocol, patients receiving room-temperature local solutions shivered slightly more frequently (46%). Jones and McLaren described visible postanesthetic shivering in 36.6% of patients after halothane and nitrous oxide anesthetics and observed that men tended to shiver four times more frequently than women.[10] Holdcroft and Hall reported that 66% of women receiving a halothane general anesthetic shivered on emergence. However, the addition of fentanyl to the anesthetic reduced the incidence to 38%.[11] Parturients receiving epidural blockade are reported to have an incidence of shivering that ranges from 31% to 64%.[8,12,13] Webb *et al.* reported shivering among 35% of women receiving room-temperature (20°C) 0.25% bupivicaine for active labor.[8] McCarroll et al.[12] reported gross shivering among 35% of patients receiving lumbar epidural for elective cesarean section, although in a similar study, Workhoven reported a 64% incidence.[13]

Quantifying postanesthetic shivering is difficult. It varies in intensity and is evanescent, making it difficult for trained observers to measure. Electromyographic (EMG) pattern analysis similar to that presented by Sessier et al.[14] may be valuable for future studies where quantification of changes in shivering patterns can be observed. EMG analysis of shivering was not available for this study. We believe that observation by trained recovery room nursing personnel, familiar with the clinical phenomenon of shivering, was adequate for the diagnosis.

Patients in the two protocols differed with respect to the temperature of intravenous solutions. There is no consensus about the effect of warm intravenous fluids on postanesthetic shivering. Workhoven reported that administration of warm crystalloid (30–33.9°C) reduced the incidence of shivering from 64% to 14% in term parturients.[13] In contrast, McCarroll *et al.* reported that administration of 2 l of 34°C crystalloid increased shivering among parturients after epidural blockade (35% vs. 55%).[12] It is unlikely that the half-liter bolus of crystalloid administered before blockade contributed to overall body temperature or to the incidence of postanesthetic shivering. The similar incidence of shivering among control patients in the meperidine protocol and warm solution protocol (40% vs. 46%) suggests little difference between groups.

Meperidine appears to stop shivering promptly during emergence from general anesthesia. Clayborn and Hirsh[5] were the first to report that meperidine, 12.5, 25, 37.5, and 50 mg, but not 6.25 mg, or saline placebo were highly effective at stopping shivering within 5 minutes and preventing its reoccurrence for 45 minutes. Pauca *et al.*[4,6] confirmed the effectiveness of meperidine, 25

TABLE 2. *Lidocaine and Catecholamine Levels*

	Nonshivering	Shivering
Lidocaine (μg/ml)	2.67 ± .98	2.07 ± .77
Epinephrine (pg/ml)		
before block	72 ± 43	93 ± 29
−15 minutes after block	445 ± 264	480 ± 299
Norepinephrine (pg/ml)		
before block	387 ± 167	468 ± 128
−15 minutes after block	638 ± 420	375 ± 293

Mean ± SD Data from nine patients who shivered and nine patients who did not shiver about 15 minutes after epidural blockade.

mg, in the control of postanesthetic shivering in 100 patients (98 general and 2 spinal). However, they found that the difference between placebo and meperidine became significant at 15 minutes. These authors observed an increased incidence of men shivering in agreement with data reported by Jones and McLaren.[10] They also reported that women stopped shivering much sooner than men after receiving meperidine, 25 mg. The results of our stury in a predominately male urologic population do not support any differences in shivering based on sex. A recent article by Macintyre *et al.*[7] confirmed that meperidine successfully terminated postanesthetic shivering. They reported that 11 of 14 (78%) patients recovering from a volatile anesthetic agent stopped shivering within 5 minutes of receiving intravenous meperidine, 25 mg.

For this study, meperidine doses reported to be effective were administered to maintain therapeutic levels during the period when shivering was expected to occur. The results showed that, when administered within 30 minutes before the blockade, meperidine was no more effective than placebo at preventing postepidural shivering. Because the goal was to prevent shivering, this experimental design differed from that of other authors who evaluated meperidine for pre-existing shivering activity. This difference may have accounted for these results. By administering the unknowns before blockade, meperidine concentration could have been "subtherapeutic" when shivering began, which could have prevented the data from reaching statistical significance.

There are other explanations for the failure of meperidine prophylaxis to prevent postepidural shivering. The mechanism of postanesthesia shivering is unknown. Loss

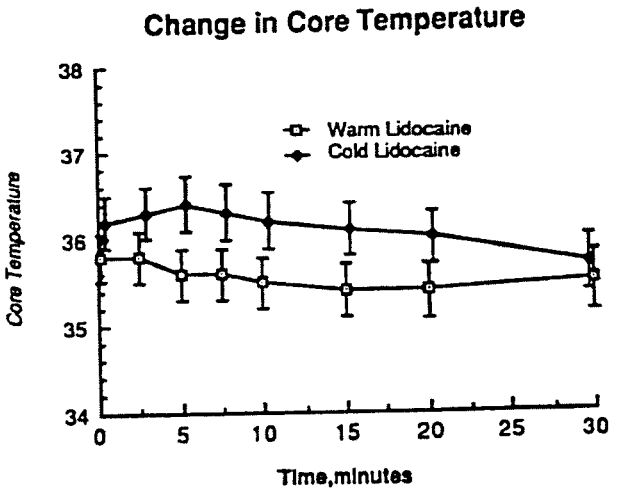

Fig. 2. Mean core (tympanic membrane) temperature in the body-temperature or room-temperature lidocaine groups during the 30-minute observation period. (All are mean ± SEM., p = 0.08 at 5 minutes.)

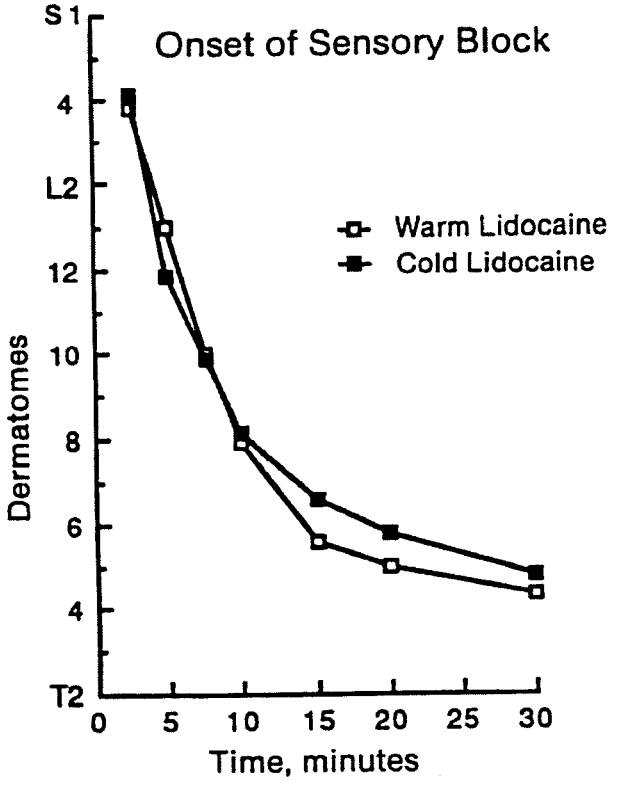

Fig. 1. Onset of sensory blockade in the body-temperature and room-temperature lidocaine groups.

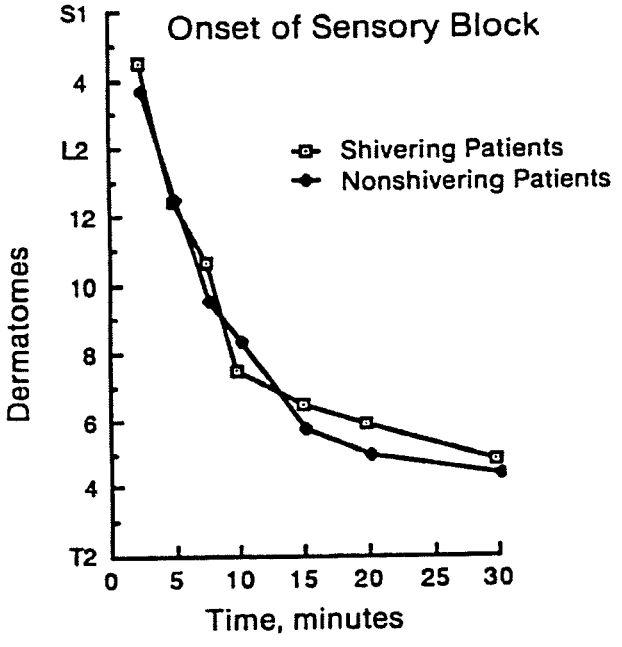

Fig. 3. Onset of sensory blockade in shivering and nonshivering patients.

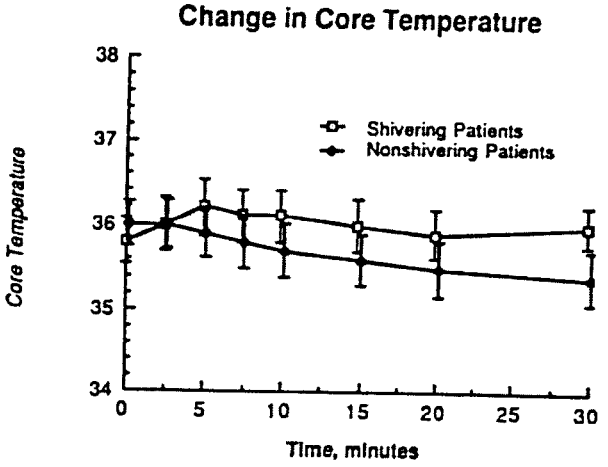

Fig. 4. Mean core (tympanic membrane) temperature in shivering and nonshivering patients during the 30-minute observation period. All are mean ± SEM.

of body heat, reduction of core temperature, activation of central heat production mechanisms, and loss of supraspinal inhibition may all play key roles.[10,11,14-17] Murphy *et al.*[16] have shown that all anesthetized monkeys shiver when body temperature decreases 2.8°C; yet, 25% of monkeys shiver with normal body temperature. Both application of radiant heat to the skin and central injection of taurine stop shivering promptly.[16] Sessler *et al.*[14] studied the EMG activity of patients shivering during recovery from general anesthesia. They observed at least two distinct EMG patterns. One was similar to that seen in cold volunteers, but the other was more similar to clonus in awake patients with spinal cord transection. They concluded that the clonus pattern represented transient loss of supraspinal inhibitory pathways and was a normal emergence phenomenon. Postanesthesia shivering may result from activation of specific heat-generating pathways or from nonspecific emergence clonus. However, shivering after epidural anesthesia could be quite different from either spontaneous clonus or cold shivering. This could explain why meperidine appears to have little effect on postepidural shivering. EMG analysis of shivering patterns after epidural blockade may be useful to differentiate mechanism.

Spinal cord thermosensitive receptors may contribute to postepidural shivering. In a study of epidural injectate temperature and postepidural shivering, Walmsley *et al.*[9] reported that injection of cold bupivacaine (4°C) decreased epidural temperature for approximately 11 minutes and resulted in shivering among 14 of 30 patients (47%) scheduled for postpartum tubal ligation. Shivering was reported to begin 3.46 to 4.36 minutes after injection. When warm bupivacaine (41°C) was injected, shivering stopped within 0.5 to 2 minutes in 4 of 8 patients (50%). It is difficult to fully explain the role of spinal cord thermosensitive receptors solely by the observations of Walmsley *et al.* Simpson and Ponte[18] examined changes in epidural temperature after injection of room-temperature bupivacaine (24°C) and reported transient drops in temperature lasting no more than 5 minutes. These observations fit with the onset of shivering seen in the study of Walmsley *et al.* but do not fit with the findings of this study. Patients in the warmed lidocaine protocol received either room-temperature (20–22°C) or body-temperature (37°C) lidocaine without any apparent effect on shivering. On the average, shivering began 13.5 minutes, not within 5 minutes, after injection. According to the work of Simpson and Ponte,[18] epidural temperatures should have equilibrated with body temperature at 13.5 minutes. These results are consistent with the work of Webb *et al.*[8] who injected epidural bupivacaine at three different temperatures: 15, 20, and 37°C. They concluded that temperature of the injectate did not influence shivering.[8] If they exist, spinal cord thermoreceptors probably play a minor role in central heat generation. It is clear from the results that shivering is not related to injectate temperature, but we can point to no single unifying theory to account for the conflicting data.

Some conclusions are relatively clear. Patients who shiver do not have blockade levels different from their nonshivering counterparts (Fig. 3), nor do they appear to be colder (Fig. 4). Patients do not shiver because of toxic levels of lidocaine from the epidural injection; lidocaine levels were not significantly different between the group that shivered and the group that did not (Table 3). There was no correlation between catecholamine levels and shivering in the group we studied, and no evidence was found to support the theory that patients shiver because of sympathetic overactivity. The data do show that serum epinephrine levels increased after administration of epidural lidocaine with epinephrine, but it may be incorrect to conclude that this rise must be wholly the result of exogenous catecholamines.

In the warmed lidocaine portion of the study, a single precalculated dose of local anesthetic was injected into the epidural space to test for onset of the blockade, body temperature, and shivering. The fact that the sensory level in 3 of 13 patients in each of the two groups did not reach T5–7 (and that one went as high as C6–7) suggests that the dosing regimen might have been more exact if it had been based on spinal column length or another index of local anesthetic spread. However, we do not believe that this had any substantive effect on our measurements or the results.

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